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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
08/905,508	08/04/1997	LALEH SHAYESTEH	02307O-06772	5513

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EXAMINER	
SITTON, JEHANNE SOUAYA	
ART UNIT	PAPER NUMBER
1634	

DATE MAILED: 07/05/2006

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	08/905,508	SHAYESTEH ET AL.	
	Examiner	Art Unit	
	Jehanne S. Sitton	1634	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *10 April 2006*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *37-39* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *37-39* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

1. Currently, claims 37-39 are pending in the instant application. Applicant's arguments have been thoroughly reviewed but are deemed insufficient to place this application in condition for allowance. The following rejections are applied to the amended claims. They constitute the complete set being presently applied to the instant Application. Response to Applicant's arguments follow where appropriate. This action is NON-FINAL.

2. The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Claim Rejections - 35 USC § 103

3. Claims 37 and 38 are rejected under 35 U.S.C. 103(a) as being unpatentable over Bonjouklian et al (hereinafter referred to as Bonjouklian; US Patent 5,378,725; 1/3/1995), in view of Arnold et al (hereinafter referred to as Arnold; Genes, Chromosomes, and Cancer, vol. 16, pages 46-54, 1996) and Volinia et al (hereinafter referred to as Volinia; Genomics, vol. 24, pp 472-477; 1994) and further in view of (in the alternative) Xiao et al (hereinafter referred to as Xiao, International Journal of Oncology; vol. 6, pp 405-411, 1995) or Skorski et al (hereinafter referred to as Skorski, Blood, vol. 86, pp 726-736, 1995).

Bonjouklian teaches and claims a method of treating PI3 kinase dependent neoplasms in mammals by administering non peptidic inhibitors (see col. 3, col. 4, table 1; col. 6, lines 49-60; and claims 1-9). Bonjouklian teaches that PI 3 kinase is an important enzyme in signal

transduction with particular implications relative to the malignant transformation of cells (col. 2, lines 22-25). Bonjouklian specifically teaches a method for treating a phosphatidylinositol 3 kinase dependent condition in a mammal, such as abnormal cell growth as found in neoplasms, such as ovarian cancer, by administering a phosphatidylinositol 3 kinase inhibiting amount of a compound as shown in cols 2, 3, and 4 (col. 6, lines 49-col. 7, line 2). Bonjouklian teaches how to determine quantity of compound, such as wortmannin (an inhibitor of PI3 kinase phosphoinositide phosphorylation), to produce a desired therapeutic effect (col. 7, especially lines 54-62). It is noted that Bonjouklian does not specifically teach detecting the presence of an amplification of PIK3CA in ovarian cancer cells from a patient, however Bonjouklian does teach treating a "PI3 kinase dependent neoplasm" and it was known in the art at the time the invention was made that the region of chromosome 3q26 comprising PIK3CA was commonly amplified in ovarian tumors as taught by Arnold (see page 49, col 2, 3q26 is increased in 42% of cases) and Volinia. Arnold specifically teaches that amplification of the 3q26-qter segment, which includes 3q26.3, suggests that the telomeric region of 3q contains one or more genes important in tumor initiation and/or progression (page 49, co. 2). Further, Volinia teaches that the catalytic p110 alpha subunit of PI 3 kinase (PIK3CA) is found in 3q26.3. Additionally, Xiao and Skorski teach that wortmannin, a known PI3 kinase inhibitor and taught by Bonjouklian as a treatment for a PI3 kinase dependent neoplasm, including ovarian cancer, was able to suppress growth of gastric cancer cells (see abstract of Xiao) and selectively inhibited the proliferation of leukemic cells (see pages 729 –730 and abstract of Skorski). Xiao teaches that growth of gastric cancer cell lines which exhibited elevated PI3 Kinase, MKN-45 and NUGC-4, was inhibited with wortmannin, while another gastric cancer cell line MKN-28, which did not exhibit such elevated

PI 3 Kinase, was more resistant to wortmannin (see abstract, page 407, col. 2, first para, page 409, col 1 and 2). Further, Xiao teaches that the activation of PI-3 kinase appears to be required for oncogenic growth of these cells (see abstract). Skorski teaches that wortmannin inhibited the growth of leukemic cells (CML) which require PI 3 kinase for proliferation (see abstract, page 731, col. 2, lines 20-24).

Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made detect amplification of PIK3CA in ovarian cancer cells in a patient and to use the PI3 kinase inhibitor wortmannin to treat ovarian cancer as taught by Bonjouklian, because Arnold teaches that such region was commonly amplified in ovarian tumors and Volinia teaches that PIK3CA is found in 3q26.3. Therefore, from the combined teachings of Volinia and Arnold, the ordinary artisan would been taught that ovarian cancer tumors would include those that had the region 3q26 amplified, including 3q26.3, suggesting that the telomeric region of 3q contains one or more genes important in tumor initiation and/or progression, as taught by Arnold, and that PIK3CA was found in the same region, as taught by Volinia. Given that Bonjouklian teaches treatment of PI3 kinase dependent neoplasms, such as ovarian cancer, the ordinary artisan would have been motivated to include ovarian tumors which were characterized by the amplification PIK3CA in the method of Bonjouklian because it was known in the art that PIK3CA was found at 3q26.3 and Xiao teaches that wortmannin, a known PI3 kinase inhibitor and taught by Bonjouklian as a treatment for a PI3 kinase dependent neoplasm, including ovarian cancer, was able to suppress growth of gastric cancer cells while Skorski teaches that wortmannin selectively inhibited the proliferation of leukemic cells (see pages 729 –730 and abstract of Skorski). Xiao teaches that growth of gastric cancer cell lines which exhibited

elevated PI3 Kinase, MKN-45 and NUGC-4, was inhibited with wortmannin, while another gastric cancer cell line MKN-28, which did not exhibit such elevated PI 3 Kinase, was more resistant to wortmannin (see abstract, page 407, col. 2, first para, page 409, col 1 and 2). The ordinary artisan would have had a reasonable expectation of success that wortmannin, as taught by Bonjouklian, would be an effective inhibitor of the pathological proliferation of ovarian tumor cells with amplification of PIK3CA because wortmannin was known to inhibit growth of different cancerous cells which had elevated PI 3 kinase activity and were PI 3 Kinase dependent as taught by Xiao and Skorski.

Response to Arguments

4. The response traverses the rejection. The response asserts that although Arnold states that amplification of 3q26.3 suggests that the regions may contain one or more genes important for tumor initiation, Arnold states that that no candidate oncogenes are known in this region and Volinia's disclosure of PIK3CA at 3q26.3 predates Arnold's work. The response concludes that Arnold therefore provides evidence that one of skill in the art prior to applicants disclosure could not have reasonably expected that PIK3CA would be an important target for treatment in ovarian cancer. This argument has been thoroughly reviewed but was found unpersuasive. In this case, although Arnold does not teach any candidate genes known in the region, it was known in the art at the time the invention as made that PI3 kinase was localized to this region of chromosome 3. Volinia specifically teaches that PIK3CA is found in the region that Arnold teaches is amplified in ovarian cancers. This knowledge was generally available to one of ordinary skill in the art at the time the invention was made. Further, Bonjouklian teaches that inhibitors of this very gene

can be used to inhibit cell growth in PIK3 dependent neoplasms. This as well as the teachings of Xiao and Skorski, that inhibition of the 110 kd subunit by wortmannin inhibits PI 3 kinas activity which is elevated in the cancer cells taught by Xiao or required for proliferation of the CML cells taught by Skorski, provides the requisite reasoning, motivation, and expectation of success to treat ovarian cancers characterized by amplification of PIK3CA with PI3 kinase inhibitors.

The response further asserts that no evidence is provided to support the proposition that one of ordinary skill would expect that cells in which 3q26 and or 3q26-qter [was amplified] would have elevations in PI3 kinase activity. This argument has been thoroughly reviewed but was found unpersuasive. The expectation that amplification in PIK3CA would lead to elevated levels or PIK3CA dependent cancer cell proliferation is provided by Bonjouklian which teaches treatment of PI3 kinase dependent neoplasms such as ovarian cancers and that PI3 kinase is an important enzyme in signal transduction with particular implications relative to the malignant transformation of cells, Xiao and Skorski which teach inhibition of cancer cell growth with a PI 3 kinase inhibitor in cancer cells exhibiting elevated PI 3 kinase and PI 3 kinase dependent proliferation. Further, the combined teachings of Arnold and Volinia (Arnold which teaches that the 3q26 region is amplified in 42% of ovarian cancers and Volinia which teaches the localization of PIK3CA to 3q26.3) provide the evidence that the chromosomal region which contains PIK3CA is amplified in 42% of ovarian cancers. Although the response questions this and states "if the Examiner's supposition were true then every gene contained within a chromosomal region that was amplified in cancer would be expected to be overexpressed", the response provides no reasoning or evidence to contradict the rejection. The rejection is not being applied to "any" amplified gene but rather to one whose activity was known, in the prior art at

the time the invention was made, to be correlated with cancer cell proliferation. One of ordinary skill would reasonably expect a correlation based on teachings of the cited prior art of Bonjouklian, Skorski, and Xiao which provides multiple examples of PI3 kinase dependent cell proliferation and which provides evidence of involvement of PI3 kinase in cancer cell proliferation. The instant rejection is not simply based on the teachings of Volinia and Arnold. Additionally, it is noted that attorney's arguments cannot take the place of evidence. As set forth in the MPEP section 2145: "The arguments of counsel cannot take the place of evidence in the record. *In re Schulze*, 346 F.2d 600, 602, 145 USPQ 716, 718 (CCPA 1965); *In re Geisler*, 116 F.3d 1465, 43 USPQ2d 1362 (Fed. Cir. 1997)".

For these reasons, the rejection is maintained.

5. Claims 37 and 38 are rejected under 35 U.S.C. 103(a) as being unpatentable over Bonjouklian in view of Daneshvar (Daneshvar et al; American Journal of Human Genetics, (1996) Vol. 59, No. 4 SUPPL., pp. A65, November 1996) and further in view of, in the alternative, Xiao or Skorski.

Bonjouklian teaches and claims a method of treating PI3 kinase dependent neoplasms in mammals by administering non peptidic inhibitors (see col. 3, col. 4, table 1; col. 6, lines 49-60; and claims 1-9). Bonjouklian teaches that PI 3 kinase is an important enzyme in signal transduction with particular implications relative to the malignant transformation of cells (col. 2, lines 22-25). Bonjouklian specifically teaches a method for treating a phosphatidylinositol 3 kinase dependent condition in a mammal, such as abnormal cell growth as found in neoplasms, such as ovarian cancer, by administering a phosphatidylinositol 3 kinase inhibiting amount of a

compound as shown in cols 2, 3, and 4 (col. 6, lines 49-col. 7, line 2). Bonjouklian teaches how to determine quantity of compound, such as wortmannin (an inhibitor of PI3 kinase phosphoinositide phosphorylation), to produce a desired therapeutic effect (col. 7, especially lines 54-62). It is noted that Bonjouklian does not specifically teach detecting the presence of an amplification of PIK3CA in ovarian cancer cells from a patient, however Bonjouklian does teach treating a "PI3 kinase dependent neoplasm". Further, Danesvhar teaches that the catalytic subunit of PI3 kinase maps to a region of chromosome 26 which is amplified in ovarian cancer. Danesvhar teaches that the gene was increased in copy number in all tumor samples and cell lines tested and showed increased expression by immunohistochemistry in tumor cell lines (see abstract). Additionally, Xiao and Skorski teach that wortmannin, a known PI3 kinase inhibitor and taught by Bonjouklian as a treatment for a PI3 kinase dependent neoplasm, including ovarian cancer, was able to suppress growth of gastric cancer cells (see abstract of Xiao) and selectively inhibited the proliferation of leukemic cells (see pages 729 –730 and abstract of Skorski). Xiao teaches that growth of gastric cancer cell lines which exhibited elevated PI3 Kinase, MKN-45 and NUGC-4, was inhibited with wortmannin, while another gastric cancer cell line MKN-28, which did not exhibit such elevated PI 3 Kinase, was more resistant to wortmannin (see abstract, page 407, col. 2, first para, page 409, col 1 and 2). Further, Xiao teaches that the activation of PI-3 kinase appears to be required for oncogenic growth of these cells (see abstract). Skorski teaches that wortmannin inhibited the growth of leukemic cells (CML) which require PI 3 kinase for proliferation (see abstract, page 731, col. 2, lines 20-24).

Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made detect amplification of PIK3CA in ovarian cancer cells in a patient

and to use the PI3 kinase inhibitor wortmannin to treat ovarian cancer as taught by Bonjouklian, because Daneshvar teaches that the PIK3CA was commonly amplified in ovarian tumors and showed increased expression by immunohistochemistry in tumor cell lines. Given that the Bonjouklian patent is directed to treatment of PI3 kinase dependent neoplasms, such as ovarian cancer, the ordinary artisan would have been motivated to include ovarian tumors which were characterized by the amplification PIK3CA in the method of Bonjouklian because it was known in the art that PIK3CA was increased in copy number in ovarian cancer cells. The ordinary artisan would have had a reasonable expectation of success that wortmannin, as taught by Bonjouklian, would be an effective inhibitor of the pathological proliferation of ovarian tumor cells with amplification of PIK3CA because wortmannin was known to inhibit growth of different cancerous cells which had elevated PI 3 kinase activity and were PI 3 Kinase dependent as taught by Xiao and Skorski.

6. Claim 39 is rejected under 35 U.S.C. 103(a) as being unpatentable over Bonjouklian, in view of Arnold and Volinia, and further in view of Xiao or Skorski, as applied to claims 37 and 38 above, and further in view of Powis (Powis et al; International Journal of Pharmacognosy, vol. 33, pages 17-26, 1995).

The teachings of Bonjouklian, Volinia, Arnold, Xiao and Skorski are set forth above. Bonjouklian & Arnold & Volinia in view of Xiao or Skorski do not teach the PI 3 kinase inhibitor LY294002 for the inhibition of the pathological proliferation of ovarian cancer cells, although Xiao does teach that LY294002 is a PI 3 kinase inhibitor. However, Powis teaches that LY294002 is a selective PI 3 kinase inhibitor (page 20, col. 1, last sentence of first full para).

Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to use LY294002 to inhibit PI 3 kinase activity in the method of Bonjouklian & Arnold & Volinia in view of Xiao or Skorski. The ordinary artisan would have been motivated to use LY294002 to inhibit PI 3 kinase activity in the method of Bonjouklian & Arnold & Volinia in view of Xiao or Skorski because it was known to be a selective inhibitor of PI 3 kinase activity as taught by Powis. The ordinary artisan would have had a reasonable expectation of success that LY294002 could be used to inhibit the pathological proliferation of ovarian cancer cells in the method of Bonjouklian & Arnold & Volinia in view of Xiao or Skorski because LY294002 was known to be an effective inhibitor of PI 3 kinase.

7. Claim 39 is rejected under 35 U.S.C. 103(a) as being unpatentable over Bonjouklian, in view of Arnold and Volinia, and further in view of Xiao or Skorski, as applied to claims 37 and 38 above, and further in view of June (US Patent 6632789).

The teachings of Bonjouklian, Volinia, Arnold, Xiao and Skorski are set forth above. Bonjouklian & Arnold & Volinia in view of Xiao or Skorski do not teach the PI 3 kinase inhibitor LY294002 for the inhibition of the pathological proliferation of ovarian cancer cells, although Xiao does teach that LY294002 is a PI 3 kinase inhibitor. However, June teaches that LY294002 is a preferred PI 3 kinase inhibitor (col. 5, lines 60-62) and teaches inhibiting a response, such as proliferation, by a T cell, using LY294002 (claims 1-19). Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to use LY294002 to inhibit PI 3 kinase activity in the method of Bonjouklian & Arnold & Volinia in view of Xiao or Skorski. The ordinary artisan would have been motivated to use

LY294002 to inhibit PI 3 kinase activity in the method of Bonjouklian & Arnold & Volinia in view of Xiao or Skorski because it was known to be a preferred inhibitor of PI 3 kinase activity as taught by June. The ordinary artisan would have had a reasonable expectation of success that LY294002 could be used to inhibit the pathological proliferation of ovarian cancer cells in the method of Bonjouklian & Arnold & Volinia in view of Xiao or Skorski because LY294002 was known to be an effective inhibitor of PI 3 kinase activity.

8. Claim 39 is rejected under 35 U.S.C. 103(a) as being unpatentable over Bonjouklian, in view of Arnold and Volinia, and further in view of Xiao or Skorski, as applied to claims 37 and 38 above, and further in view Lavin (Lavin et al; Experientia, vol. 52, pages 979-994, 1996).

The teachings of Bonjouklian, Volinia, Arnold, Xiao and Skorski are set forth above. Bonjouklian & Arnold & Volinia in view of Xiao or Skorski do not teach the PI 3 kinase inhibitor LY294002 for the inhibition of the pathological proliferation of ovarian cancer cells, although Xiao does teach that LY294002 is a PI 3 kinase inhibitor. However, Lavin teaches that LY294002 is an effective PI 3 kinase inhibitor and abrogated the ability of NGF to prevent apoptosis in PC-12 cells, suggesting one important role of PI 3 kinase is to ensure cell survival by preventing apoptosis (986, col. 2, lines 18-25). Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to use LY294002 to inhibit PI 3 kinase activity in the method of Bonjouklian & Arnold & Volinia in view of Xiao or Skorski. The ordinary artisan would have been motivated to use LY294002 to inhibit PI 3 kinase activity in the method of Bonjouklian & Arnold & Volinia in view of Xiao or Skorski because it was known to be a selective inhibitor of PI 3 kinase and cell growth as taught by Lavin. The

ordinary artisan would have had a reasonable expectation of success that LY294002 could be used to inhibit the pathological proliferation of ovarian cancer cells in the method of Bonjouklian & Arnold & Volinia in view of Xiao or Skorski because LY294002 was known to be an effective inhibitor of PI 3 kinase and to abrogate the ability of a growth factor to prevent apoptosis.

Response to Arguments

9. The response traverses the rejections of claim 39 under 35 USC 103 for the reasons made of record with regard to the rejection of claims 37 and 38, and states that the additional references merely teach that LY294002 is a PI3Kinase inhibitor. This argument has been thoroughly reviewed but was not found persuasive for the reasons made of record above.

10. Claim 39 is rejected under 35 U.S.C. 103(a) as being unpatentable over Bonjouklian in view of Daneshvar and further in view of Xiao or Skorski, as applied to claims 37 and 38 above, and further in view of, in the alternative, Powis, or June, or Lavin.

The teachings of Bonjouklian, Daneshvar, Xiao and Skorski are set forth above. Bonjouklian & Daneshvar in view of Xiao or Skorski do not teach the PI 3 kinase inhibitor LY294002 for the inhibition of the pathological proliferation of ovarian cancer cells, although Xiao does teach that LY294002 is a PI 3 kinase inhibitor.

Powis teaches that LY294002 is a selective PI 3 kinase inhibitor (page 20, col. 1, last sentence of first full para).

June teaches that LY294002 is a preferred PI 3 kinase inhibitor (col. 5, lines 60-62) and teaches inhibiting a response, such as proliferation, by a T cell, using LY294002 (claims 1-19).

Lavin teaches that LY294002 is an effective PI 3 kinase inhibitor and abrogated the ability of NGF to prevent apoptosis in PC-12 cells, suggesting one important role of PI 3 kinase is to ensure cell survival by preventing apoptosis (986, col. 2, lines 18-25).

Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to use LY294002 to inhibit PI 3 kinase activity in the method of Bonjouklian & Daneshvar in view of Xiao or Skorski. The ordinary artisan would have been motivated to use LY294002 to inhibit PI 3 kinase activity in the method of Bonjouklian & Daneshvar in view of Xiao or Skorski because it was known to be an inhibitor of PI 3 kinase activity as taught by Powis, June, or Lavin. The ordinary artisan would have had a reasonable expectation of success that LY294002 could be used to inhibit the pathological proliferation of ovarian cancer cells in the method of Bonjouklian & Daneshvar in view of Xiao or Skorski because LY294002 was known to be an effective inhibitor of PI 3 kinase activity.

Conclusion

11. No claims are allowable over the cited prior art.

12. Any inquiry concerning this communication or earlier communications from the examiner should be directed to examiner Jehanne Sitton whose telephone number is (571) 272-0752. The examiner can normally be reached Monday-Thursday from 8:00 AM to 5:00 PM and on alternate Fridays.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla, can be reached on (571) 272-0735. The fax phone number for this Group is (571) 273-8300.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to (571) 272-0547.

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Jehanne Sitton
Primary Examiner
Art Unit 1634
6/22/06